IN THE UNITED STATES PATENT AND TRADEMARK OFFICE

In re Application of: CAROL CARTER, ET AL.	)	Confirmation No: 6642
	)	
Application No.: 10/666,997	)	Group Art Unit: 1648
	)	
Filed: SEPTEMBER 18, 2003	)	Examiner: HUMPHREY, LOUISE

For: TSG101 AS INHIBITOR OF HIV PRODUCTION

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P.O. Box 1450
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SUPPLEMENTAL SUBMISSION ON APPEAL

Sir:

Applicants, Appellants herein, supplement their Appeal Brief and Reply Brief with the article submitted herewith, newly printed in Bioorganic and Medicinal Chemistry Letters. *Liu et al., Bioorganic & Medicinal Chemistry Letters* 20, 318 – 321 (2010) confirm in fact that the claims pending on appeal, directed to a method of inhibiting viral budding in HIV infected cells, is enabled by the specification which the Examiner agreed allows one to identify all PTAP – containing peptides that bind to TSG101 and inhibit binding between TSG101 and the HIV Gag protein to interfere with the viral life cycle and budding. An appropriate Appendix is submitted herewith.

Appellants' broadest claim on appeal is Claim 93:

> A method of inhibiting human immunodeficiency virus (HIV) particle generation comprising administering to cells suspected of being infected with HIV an amount of a compound which inhibits binding between tumor susceptibility gene (TSG101) protein and HIV Gag polypeptide, wherein said compound is a peptide comprising a PTAP motif.

The sole rejection of this claim is for lack of enablement – it being the Examiner's opinion that notwithstanding the disclosure, which the Examiner acknowledges allows one of skill in the art as of its filing date to identify all PTAP comprising peptides that interfere with binding between TSG101 and HIV Gag and therefore inhibit viral budding, the lack of any working examples demonstrates a lack of compliance with 35 U.S.C. §112, first paragraph.

The article submitted herewith demonstrates that in fact linear and cyclic peptides which comprise the PTAP motif (see, p. 318, left-hand column) when administered to HIV infected cells inhibit viral particle release (see, the abstract, and first paragraph, page 318). Given the ability to identify the appropriate PTAP binding peptides, this submitted article demonstrates that not only can viral particle propagation be inhibited by administration of the same, but that routine chemistry can allow one to increase uptake and inhibition of viral particle release. Clearly, given Applicants' disclosure of an assay that allows identification of PTAP-comprising peptides that will inhibit viral release, and the post filing confirmation that in fact such peptides can be used to achieve reduction in HIV particle generation, there can be no question that the claims on appeal are enabled.

Appellants apologize for the submission of this article after the normal filing cycle in this appeal. It was only recently released and brought to the attention of the Applicants. It is of

particular significance that the work reported in the article submitted herewith was conducted and reported independent of Applicants and the real parties-in-interest therefore.

Date: May 25, 2010

Respectfully submitted,

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I. EVIDENCE APPENDIX

The following is a list of references relied upon by Appellant in this appeal, along with a statement setting forth where in the record that evidence was entered by the Appellant. A copy of the evidence is provided herewith.

REFERENCE	LOCATION IN THE RECORD
Liu et al., Bioorganic & Medicinal Chemistry Letters 20, 318-321 (2010)	Appellants' Supplemental Submission on Appeal filed on May 25, 2010.
Article confirming that in fact the claims pending on this appeal are enabled by the disclosed method which the Examiner agreed allows one to identify all PTAP – containing peptides that bind to TSG101 and inhibit binding between TSG101 and the HIV Gag protein to interfere with the viral life cycle and budding.	